old pleuritic adhesions. A small amount of fluid in pericardium. Heart of normal size. No evidences of atheroma of aorta or coronaries. Valves all normal. Abdomen: viscera normal. Brain: cranial bones thicker than normal; no traces of sutures; two depressions by the side of the sagittal line in front. Right half of cranium has less capacity than the left. When the dura was incised a clear serum escaped; the dura was thickened; arachnoid and pia pallid and infiltrated with serum. Pia readily came away from cortex beneath. No atheroma of Sylvian artery or other vessels. The brain substance was pale, and of normal consistency. Nothing abnormal was discovered in the nerve centers as far as the naked eye appearances are concerned. The microscopic findings are as follows: the dendrites showed a rosary-like series of swellings, varicosities. The fibers in the white substance also exhibited varicosities and atrophy. The neuroglia was in general atrophic. In the cerebellum the pigment in the nerve cells was much diminished. In the cord, the ganglionic elements exhibited a breaking up of the pigment. This chromatolysis was marked in all the cells of the medulla. The entire nervous system whether encephalon, spinal cord, peripheral nerves. etc., showed the same type of degeneration. This degeneration was based upon the anemic condition of the brain, especially in the Rolandic region and foot of the third frontal convolution on both sides, in which regions the epileptogenous zone appears to be seated.

CLARK.

EIN FALL VON TRAUMATISCHER SPÄTAPOPLEXIE (A Case of late Traumatic Apoplexy) J. Mazurkiewicz (Jahrbücher für Psychiatrie und Neurologie, Vol. xix, 19th Year, p. 551).

Case of a man who received a trauma from a fall. No symptoms followed immediately upon the accident. A few weeks later the patient fell again. Following this there developed headaches, somnolence, speech disturbance, paralysis. An examination made five days after the second fall showed no paresis, ataxia, or sensory disturbance. No mental involvement. The speech however was monotonous and heavy. There developed in the course of a week aphasia with, however, a complete comprehension of words, difficulty in deglutition, and gradually increasing right-sided paralysis with spastic condition of lower extremities. Under increasing stupor and inability to take nourishment, the patient died. Autopsy: Left lateral ventricle is full of blood originating from a hemorrhagic focus in the thalamus opticus, which had broken through into the ventricle. Third ventricle also contained fluid blood. Hehorrhagic focus in left side of pons. These findings are typical of late apoplexy and confirm the opinion of Michel that the site of predilection in late traumatic apoplexy is first the base of the brain, then the walls of the lateral ventricle, the aqueduct of Sylvius and the fourth ventricle. The diagnosis of this condition can be very difficult, even impossible, especially when the clinical history cannot be obtained. Tumor, abscess, encephalitis are of differential diagnostic importance. When symptoms pointing to a break into the ventricles are present a lumbar puncture may show the presence of a hemorrhagic fluid, as was found in this case.

SCHWAB.

TOXICITÉ DE LA SUEUR DES EPILEPTIQUES (Toxicity of the Sweat of Epileptics), Mairet and Ardin-Delteil (Soc. de Biol. Dec. 14. 1900).

These authors have recently studied the toxicity of the normal sweat and have repeated the experiments with epileptic sweat. In the first series of ten cases the epileptic sweat exhibited no difference in comparison with normal sweat, save that the digestive tube was more affected than in the latter case, while the urine was also more frequently voided. Fatalities were not more commonly produced. In a second series, however, the epileptic sweat proved fatal in every one of eight cases, and in as small amounts as 137 c. c., 151 c. c., etc. It was exceptional, however, for convulsions to develop. The autopsy findings showed much inconstancy. To explain such radical differences in results in these two groups of cases, the authors state that in the first series, the sweat used was interparoxysmal, while in the second series it was taken either at the onset of the attack or very soon afterward.

To sum up, the interparoxysmal sweat is normal sweat, and the paroxysmal sweat is toxic and lethal in action. The toxicity is soon lost after the attack. This toxic property of sweat is genuine and not due to want of isotony with the blood serum of the rabbit. For that matter, the isotony is often almost perfect. Thus freezing point of the paroxysmal sweat runs from 40 to 56, while the interparoxysmal sweat is about 40.

Referring to the prior article by these authors on the normal sweat (Compes Rend. Soc. de Biol., Nov. 23, 1900), a description for their method of collecting the sweat is given. The patient is placed in a coffin-shaped oven, his head alone being outside of the apparatus. The latter is heated by a current of hot air; the skin was previously ascepticised. The sweat during the baking process ran down into a trough whence it gravitated to a sterile collecting vessel. In some cases the authors collected as much as 800 c. cm. of sweat from a single individual. CLARK.

UNFALL UND EPILEPSIE (Epilepsy and Accident). F. Bahr (Monatssch. f. Unfallheilkunde, Oct. 15, 1900).

The literature of traumatic epilepsy of peripheral origin is a meager one. Cases are confined almost wholly to the results of direct injury to the skull and brain. There is hardly anything on the subject of epilepsy as a result of peripheral wounds. In the Franco-German war, out of 77,461 wounded (not including cases which resulted fatally) there were 17 cases of traumatic reflex epilepsy. Despite this infrequency the author believes it well to call attention to the subject. He has been able to get notes of three cases in which an unmistakeable connection can be shown between accident and epilepsy.

Case I. Male, aged 44; his history negative. He wrenched his right shoulder while carrying a drain pipe. The clavicle of the same side also was fractured; the whole right half of thorax was sensitive. Traumatic hemoptysis. After his injuries had healed, he complained of spells of forgetfulness, unconsciousness, vertigo, etc. About a year after the injury, he developed typical epileptic attacks. Case II. Male, aged 44; history before injury was quite negative. His accident consisted of a fall which apparently caused a contusion of the lung. He became subject to epileptic attacks. Case III. Male, aged 56; his history was negative; he had a fall from a ladder. He developed epileptic attacks which appeared to be called forth by injury to the left lower extremity.

All these cases were in middle-aged men; it is rare for epilepsy to begin after the age of 20 years. The coincidence is a much more